inch to the outer side of the crest of the tibia. Often both bones can be treated through this incision. Care must be taken not to injure the anterior or the posterior tibial vessels and nerves. If a separate incision is necessary to expose the fibula, this may be made at any point along the line of the fibula from the head to the external malleolus, entering the space between the extensor longus digitorum on the inner side and the peroneal muscles on the outer side, being careful not to injure the musculocutaneous nerve, or, if the operation is near the head of the fibula, the peroneal nerve. Often the position of the incision is determined by a wound already existing in the soft parts. In one case the styloid process and insertion of the biceps were broken from the fibula by muscular action (wrestling). Suture with kangaroo tendon gave a good result.

LEUKEMIC BLOOD PICTURE IN A CASE OF FRACTURE OF THE ANKLE.

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Reports upon the blood findings in cases of fracture are so scarce that it is difficult to form a definite opinion of the frequency with which abnormalities occur. Cabot[1] states that a study of thirty-eight cases, by himself, Hubbard, and Blake, of which fifteen were complicated, showed nothing abnormal. From the figures given, however, it is clear that in a certain percentage of the cases the leukocytes were increased. Ewing and Da Costa give no original observations, and Grawitz also makes no special mention of the findings in fractures. A survey of the journal literature of the past few years likewise brought no data to light. Hastings writes me that in two cases he has seen "myelemia," with myelocytes between 3 and 7 per cent., the one being a severe crush of the thigh (and femur), and the other one of the forearm, with no break in the skin and no evidence of infection.

In view of these meager data the following case report may not be out of place: The patient was a colored man, aged forty-five years, who was admitted, on October 18, 1905, to Dr. Johnson's service at the Maryland General Hospital, with a compound fracture of the right ankle. Careful questioning did not bring out any information which would suggest that the patient had been the subject of myeloid leukemia previous to the accident. He had noticed no lump in the abdomen and had been in his usual strength up to that time.

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[1] Pathology of the Blood, 5th ed. p. 400.

Differential counts were made on October 31, November 2, 4, and 6, and showed an increase of the polynuclear neutrophiles (79 to 83 per cent.) and low eosinophile values, but no myelocytes or other unusual condition.

Cellulitis developed, and on November 15 the leg was amputated in the lower third. The day before the amputation the leukocytes were much increased (50,000 estimated) and a differential count revealed a state of affairs which was typical of myeloid leukemia: small lymphocytes, 3.0 per cent.; large mononuclears, 2.5 per cent.; polynuclear neutrophiles, 56.2 per cent.; eosinophiles, 6.2 per cent.; mast cells, 17.5 per cent.; neutrophilic myelocytes, 15.0 per cent.; eosinophilic myelocytes, 1.2 per cent. While counting, 400 leukocytes, 22 normoblasts and several megaloblasts were seen.

These findings, which were first reported by the senior students who had charge of the hematological observations in the case, were verified by my assistant, Dr. Langley, and myself. The spleen, which had not been examined before, was now readily palpable at the costal border.

Unfortunately no blood specimens were taken again until November 21. At that time the leukemic blood picture was much less pronounced. The leukocytes had diminished considerably in number, and myelocytes, while still present, were less numerous. The mast cells had fallen to 4 per cent., and only an occasional normoblast was seen. After that the myelocytes gradually disappeared, and when the patient was discharged the blood picture was entirely normal.

An examination on April 23, 1906, showed 30 per cent. of small lymphocytes, 10 per cent. of large mononuclears, 59 per cent. of polynuclear neutrophiles, 4 per cent. of eosinophiles, and less than 1 per cent. of mast cells. There were no myelocytes and no nucleated red cells.

An examination of the bone-marrow of the amputated foot was unfortunately neglected.

To recapitulate we have a blood picture, occurring in a patient after a compound fracture of the leg, at a time when cellulitis had developed, which bears every mark that is typical of the myeloid type of leukemia. After a week it wanes, and with the recovery of the patient there is a return to normal conditions. The degree of splenic enlargement, when I first saw the patient, a few days after the discovery of the remarkable blood findings, was not at all proportionate to the blood picture, as we generally see it in myeloid leukemia, but not inconsistent with that diagnosis. At the time I was inclined to think that the case was a true leukemia and that the accident had possibly occurred during a "normal" interval. But the subsequent course of the myelemia inclined me to abandon this view. The return of the mast cells to the normal especially speaks against this possibility. Ehrlich long ago pointed out that the increase of the mast cells is the most constant and characteristic symptom of myeloid leukemia, and I have pointed out at another place that this increase will persist even though the spleen may return to normal size and the blood picture clear up otherwise. Nevertheless, we must assume a *vis a tergo*, and notwithstanding the well-known effect of septic infection upon the character of the leukocytosis under ordinary circumstances, I believe that this factor must be viewed directly or indirectly as a causal element after all. It is known that in osteomyelitis, myelocytes may appear in the circulating blood in large numbers, although this does not occur in every case. An increase of the mast cells and the simultaneous occurrence of nucleated red cells, on the other hand, have not been observed. I can imagine, however, that the findings may differ with a varying localization of the inflammatory process, and that extensive involvement of red marrow may give rise to a different picture than would result if the fatty marrow were largely affected. This idea would, in a manner, presuppose the origin of the marrow elements from the affected structures, which would, on the face of it, seem unlikely. On the other hand, one might imagine that the extensive destruction of red marrow in the injured bones, especially after development of the infection, might lead to a stimulation of the red bone-marrow at large and a resultant functional hyperactivity and the mechanical discharge of bone-marrow elements into the circulation. In this sense the temporary myelemia in my case could be regarded as the leukocytic counterpart to the "crises" affecting the red cells, which are occasionally observed in certain types of severe anemia. This view is the one to which I incline. The case is certainly unique and is reported with the idea of bringing out records of similar cases, which have not yet been reported in the literature.

THE LUYS URINE SEPARATOR.

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The history of separators for the intravesical separation of the urine from the two kidneys dates from 1890, when Lambotte,[1] of Brussels, first introduced an instrument which proved unsuccessful. In 1897 Neumann[2] was also unsuccessful in producing a separator

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[1] Etude sur la taille du rein. Jour. de méd. de chir. et de phar., Bruxelles, Septembre, Octobre, et Novembre, 1890.

[2] Ein einfache Methode den Urine beider Nieren bei Weibe gesondert aufzufangen. Deutsch. med. Woch., October 21, 1897.